WEST NILE VIRUS DISEASE

DISEASE REPORTING

In Washington

West Nile virus (WNV) disease is a notifiable condition reported as Arboviral (arthropod-borne viral) Disease.

WNV disease, caused by infection with an arthropod-borne flavivirus was first reported in the Western Hemisphere in 1999 during an outbreak in New York City. Since then, WNV has spread throughout the United States (U.S.) infecting reservoir birds, vector mosquito species, horses, and humans. During 2003, there were 9,862 reported human cases, including 264 fatalities, nationwide. Although a few cases of WNV disease have been reported in Washington State residents who were exposed to infected mosquitoes while traveling outside the state, as of July 2004, no human cases of WNV disease have been acquired within Washington State.

Purpose of reporting and surveillance

- To assist in the detection of cases, and prevent disease transmission.
- To distinguish WNV infection acquired locally from those related to travel.
- To monitor the epidemiology of WNV disease in Washington State and target mosquito control measures.
- To identify emerging infections in Washington State.

Reporting requirements

- Health care providers: notifiable to Local Health Jurisdiction within 3 work days.
- Hospitals: notifiable to Local Health Jurisdiction within 3 work days.
- Laboratories: isolation of WNV or detection of viral antibody or nucleic acid notifiable to Local Health Jurisdiction in which within 2 work days.
- Local Health Jurisdictions: notifiable to DOH Communicable Disease Epidemiology within 7 days of case investigation completion or summary information required within 21 days.

Clinical criteria for diagnosis

The majority of patients with WNV infections are asymptomatic; approximately 20% have mild self-limited illness, and less than 1% develop severe neurological disease. Symptoms can include fever, weakness, gastrointestinal symptoms, change in mental status, headache, myalgias, rash, lymphadenopathy, meningismus, cranial nerve palsies, paresis, flaccid paralysis, abnormal reflexes, seizures, movement disorders, and coma.

WNV infections are classified as either neuroinvasive or non-neuroinvasive, according to the following criteria:

Neuroinvasive disease requires the presence of fever and ***at least one*** of the following, as documented by a physician and in the absence of a more likely clinical explanation:

- Acutely altered mental status (e.g., disorientation, obtundation, stupor, or coma), or
- Other acute signs of central or peripheral neurologic dysfunction (e.g., paresis or paralysis, nerve palsies, sensory deficits, abnormal reflexes, generalized convulsions, or abnormal movements), or
- Pleocytosis (increased white blood cell concentration in cerebrospinal fluid [CSF]) associated with illness clinically compatible with meningitis (e.g., headache or stiff neck).

Non-neuroinvasive disease requires, at minimum, ***all*** of the following:

- Presence of documented fever, as measured by the patient or clinician, and
- Absence of neuroinvasive disease (as described above), and
- Absence of a more likely clinical explanation.

Laboratory criteria for diagnosis

- Fourfold or greater change in serum antibody titer, or
- Isolation of virus from, or demonstration of viral antigen or genomic sequences in tissue, blood, cerebrospinal fluid (CSF), or other body fluid, or
- Virus-specific immunoglobulin M (IgM) antibodies demonstrated in CSF by antibody-capture enzyme immunoassay (EIA), or
- Virus-specific IgM antibodies demonstrated in serum by antibody-capture EIA and confirmed by demonstration of virus-specific serum immunoglobulin G (IgG) antibodies in the same or a later specimen by another serologic method (e.g., plaque reduction neutralization or hemagglutination inhibition).

Case definition

- Probable: a clinically compatible case occurring during a period when arboviral transmission is likely with the following supportive serology: 1) a single or stable but elevated titer (≤ twofold change) of virus-specific serum antibodies; or 2) serum IgM antibodies detected by antibody-capture EIA but with no available results of a confirmatory test for virus-specific serum IgG antibodies in the same or a later specimen.
- Confirmed: a clinically compatible case that is laboratory confirmed.

Because closely related arboviruses exhibit serologic cross-reactivity, positive results of serologic test using antigens from a single arbovirus can be misleading. In some circumstances (e.g., in areas where two or more closely related arboviruses occur, or in imported arboviral disease cases), it may be epidemiologically important to attempt to pinpoint the infecting virus by conducting cross-

neutralization tests using an appropriate battery of closely related viruses. This is essential, for example, in determining that antibodies detected against St. Louis encephalitis are not the result of an infection with West Nile (or dengue) virus, or vice versa, in areas where both of these viruses occur.

A. DESCRIPTION

1. Identification

Most WNV infections are asymptomatic; about 20% of infected persons will have fever and other symptoms that may persist for weeks, and <1% will develop serious neurological disease involving the brain, spinal cord and/or meninges. In the initial 1999 U.S. outbreak, more than 50% of the hospitalized patients had severe muscle weakness, and some were initially thought to have Guillain-Barré syndrome. Neuroinvasive infections usually involve meningitis and/or encephalitis with or without additional neurological features such as acute flaccid paralysis, ataxia, extrapyramidal signs, cranial nerve abnormalities, myelitis, optic neuritis, polyradiculitis. The risk of severe WNV infection and death is highest in individuals over the age of 50 years. Overall case-fatality rates range from 4-14%, but among hospitalized patients over 70 years of age, case-fatality rates are 15-29%. Increasing data suggest that long-term sequelae, including neurologic deficits, are not uncommon among survivors of severe neuroinvasive infection.

Signs and symptoms of WNV disease cannot be reliably distinguished from other neurotropic viral infections, including other domestic arboviral diseases, such as St. Louis encephalitis and Western Equine encephalitis, which are endemic in Washington State. The peak of human WNV infection in the United States is July-October. Enteroviral infections are clinically indistinguishable from WNV disease, especially in children. Viral-specific laboratory tests are needed to confirm the etiology of infection.

2. Infectious Agent

WNV is a single-stranded RNA virus of the *Flaviviridae* family (*flavivirus*) and is part of the Japanese encephalitis virus serocomplex, which contains several important human encephalitis viruses: Japanese encephalitis, St. Louis encephalitis, yellow fever, dengue, Murray Valley encephalitis, and Kunjin. Antibodies against flaviviruses may cross-react and a history of recent vaccination or travel to a flavivirus-endemic area provides pertinent epidemiological information.

3. Worldwide Occurrence

WNV was first isolated and identified in 1937 from an infected woman in the West Nile district of Uganda. Until 1999, WNV disease was found only in the Eastern Hemisphere, with wide distribution in Africa, Asia, the Middle East, and Europe. Human outbreaks occur and have been more frequent and associated with more severe neurological disease since the mid-1990's. Outbreaks in Romania, Israel, Russia, and the U.S. have involved thousands of cases with significant neurological disease.

4. Reservoir

Due to the recent introduction of WNV in the U.S., investigation of the ecology of the virus in the Western Hemisphere is ongoing. WNV has been detected in >35 North American mosquito species and >225 bird species. Corvids (e.g., crows, magpies, jays) often succumb to the infection. Mosquitoes of the genus Culex (e.g., *Culex pipiens*, *Culex restuans*, *Culex quinquefaciatus, Culex tarsalis)* are among the most competent WNV vectors in the U.S. The virus is maintained in an enzootic cycle involving birds as reservoirs and mosquito vectors. Amplification of WNV among birds and mosquitoes occurs during warm weather; the peak of viral transmission to humans and horses occurs during August and September. Humans and other mammals (primarily horses) are incidentally infected, and are not reservoirs for mosquito infection.

5. Mode of Transmission

WNV is primarily transmitted by mosquito bites. WNV disease is not transmitted by direct contact with infected humans, birds, or animals. Unusual modes of transmission include transfusion of contaminated blood products, transplantation of infected organs or tissue, from an infected woman to child during pregnancy or breastfeeding, and percutaneous injuries while working with infected animals or in a laboratory setting.

6. Incubation period

Two to fifteen days.

7. Period of communicability

Donated blood units are routinely tested for WNV to prevent transmission by transfusion. Mosquitoes remain infective for life, and viremic birds are the source of infection for mosquitoes. After infection, a transient viremia occurs but this is not reliably detectable in the blood or CSF of humans after onset of symptoms. Horses develop active disease but viremia is not present in high titer or for long periods; therefore, humans and horses are not sources of mosquito infection.

8. Susceptibility and resistance

Susceptibility to WNV disease increases with age, and risk for serious infection is highest among persons >50 years of age and among persons with certain immunocompromising conditions. Persistence of antibodies against WNV is probably lifelong, but further research is needed to determine whether re-infection can occur.

B. METHODS OF CONTROL

1. Preventive measures:

a. Avoid exposure to mosquitoes, especially those active during dusk and dawn.
b. Wear long sleeve shirts and long pants to avoid mosquito exposure.
c. Use DEETcontaining mosquito repellent when needed.
d. Eliminate breeding places for mosquitoes, e.g., eliminate standing water in tires, buckets, play toys, flowerpots, gutters, etc.
e. Change water twice weekly in bird baths, pet dishes, fountains, and wading pools.
f. Fix leaky outdoor faucets
g. Screen doors and windows of sleeping and living quarters.

2. Control of patient, contacts and the immediate environment:

a. Report suspected, probable, and confirmed cases to Local Health Jurisdiction.
b. Isolation: None; virus is not usually found in blood, secretions or discharges during clinical disease. Enteric precautions are appropriate until enterovirus meningoencephalitis can be ruled out.
c. Concurrent disinfection: None.
d. Quarantine: None.
e. Immunization of contacts: None.
f. Investigation of contacts and source of infection: Search for additional cases and the presence of vector mosquitoes (see B3 below).
g. Specific treatment: None.

3. Epidemic measures

a. Identification of infection among horses, birds, and humans in the community indicates the presence of viral activity in mosquito vectors and may define the areas affected. Immunization of horses does not limit spread of the virus in the community.
b. Integrated pest management techniques for controlling mosquito vectors.

4. International measures

Enforcement of international agreements designed to prevent transfer of mosquitoes by ships, airplanes, and land transport.